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NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
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NEWS	3	Jan 29	FSTA has been reloaded and moves to weekly updates
NEWS	4	Feb 01	DKILIT now produced by FIZ Karlsruhe and has a new update frequency
NEWS	5	Feb 19	Access via Tymnet and SprintNet Eliminated Effective 3/31/02
NEWS	6	Mar 08	Gene Names now available in BIOSIS
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NEWS	8	Mar 22	TRCTHERMO no longer available
NEWS	9	Mar 28	US Provisional Priorities searched with P in CA/CAplus and USPATFULL
NEWS	10	Mar 28	LIPINSKI/CALC added for property searching in REGISTRY
NEWS	11	Apr 02	PAPERCHEM no longer available on STN. Use PAPERCHEM2 instead.
NEWS	12	Apr 08	"Ask CAS" for self-help around the clock
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NEWS	15	Apr 19	US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
NEWS	16	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
NEWS	17	Apr 22	BIOSIS Gene Names now available in TOXCENTER
NEWS	18	Apr 22	Federal Research in Progress (FEDRIP) now available

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FILE 'BIOSIS' ENTERED AT 16:16:14 ON 26 APR 2002
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=> s u3 and polyadenylation
L1          65 U3 AND POLYADENYLATION

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L2           39 DUPLICATE REMOVE L1 (26 DUPLICATES REMOVED)

=> d 1-10 bib ab

L2   ANSWER 1 OF 39     MEDLINE                          DUPLICATE 1
Full Text
AN   2002111907     MEDLINE
DN   21826704   PubMed ID: 11836419
TI   The solitary long terminal repeats of ERV-9 endogenous retrovirus are
     conserved during primate evolution and possess enhancer activities in
     embryonic and hematopoietic cells.
AU   Ling Jianhua; Pi Wenhu; Bollag Roni; Zeng Shan; Keskintepe Meral; Saliman
     Hatem; Krantz Sanford; Whitney Barry; Tuan Dorothy
CS   Department of Biochemistry and Molecular Biology, School of Medicine,
     Medical College of Georgia, Augusta, GA 30912, USA.
NC   DK 15555 (NIDDK)
     HL 39948 (NHLBI)
     HL 62308 (NHLBI)
SO   JOURNAL OF VIROLOGY, (2002 Mar) 76 (5) 2410-23.
     Journal code: 0113724. ISSN: 0022-538X.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Priority Journals
OS   GENBANK-AC005202; GENBANK-AF064190; GENBANK-AF094515; GENBANK-AF139840;
     GENBANK-AF141972; GENBANK-AF141973; GENBANK-AF141975; GENBANK-AF227991;
     GENBANK-AF227992; GENBANK-AF227993; GENBANK-AF227994; GENBANK-AF227995
EM   200203
ED   Entered STN: 20020215
     Last Updated on STN: 20020317
     Entered Medline: 20020315
AB   The solitary long terminal repeats (LTRs) of ERV-9 endogenous retrovirus
     contain the U3, R, and U5 regions but no internal viral genes. They are
     middle repetitive DNAs present at 2,000 to 4,000 copies in primate
     genomes. Sequence analyses of the 5" boundary area of the erythroid
     beta-globin locus control region (beta-LCR) and the intron of the
     embryonic axin gene show that a solitary ERV-9 LTR has been stably
     integrated in the respective loci for at least 15 million years in the
     higher primates from orangutan to human. Functional studies utilizing the
     green fluorescent protein (GFP) gene as the reporter in transfection
     experiments show that the U3 region of the LTRs possesses strong
     enhancer activity in embryonic cells of widely different tissue origins
     and in adult cells of blood lineages. In both the genomic LTRs of
     embryonic placental cells and erythroid K562 cells and transfected LTRs of
     recombinant GFP plasmids in K562 cells, the U3 enhancer activates
     synthesis of RNAs that are initiated from a specific site 25 bases
     downstream of the AATAAA (TATA) motif in the U3 promoter. A second
     AATAAA motif in the R region does not serve as the TATA box or as the
     polyadenylation signal. The LTR-initiated RNAs extend through the R and
     U5 regions into the downstream genomic DNA. The results suggest that the
     ERV-9 LTR-initiated transcription process may modulate transcription of
     the associated gene loci in embryonic and hematopoietic cells.

L2   ANSWER 2 OF 39     MEDLINE
Full Text
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AN 2001555089 MEDLINE
DN 21487365 PubMed ID: 11601755
TI Genetic reshuffling reconstitutes functional expression cassettes in retroviral vectors.
AU Tabotta W; Klein D; Hohenadl C; Salmons B; Gunzburg W H
CS Institute of Virology, University of Veterinary Sciences, Vienna, Austria.
SO JOURNAL OF GENE MEDICINE, (2001 Sep-Oct) 3 (5) 418-26.
Journal code: 9815764. ISSN: 1099-498X.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200202
ED Entered STN: 20011017
Last Updated on STN: 20020222
Entered Medline: 20020221
AB BACKGROUND: A major prerequisite for the design of retroviral vectors encoding cell toxic or harmful genes is the possibility to tightly control gene expression, thus limiting activity to the relevant target cells and protecting the packaging cell used for production of recombinant viral particles. METHODS: In the present study a system was developed in which genetic reshuffling during the retroviral life cycle is exploited, allowing reconstitution of functional expression cassettes from separate elements exclusively in transduced target cells. For construction of these murine leukaemia virus (MLV)-based reconstituting viral vectors (ReCon), a promoterless inverted enhanced green fluorescent protein (EGFP) reporter gene cassette was inserted in place of the **U3** region of the 3' LTR. Subsequently, the human ubiquitin promoter was inserted in the inverse orientation into the R/U5 border of the 5' LTR of the vector. RESULTS: PA317 packaging cells stably transfected with ReCon vectors were established and EGFP expression was analysed by fluorescence-activated cell sorting (FACS). After detection of low-level background expression, an additional **polyadenylation** signal was introduced in antisense orientation into the 3' LTR at the R/U5 border to prevent accidental read-through transcription from neighbouring cellular promoters. Virus-containing cell culture supernatants were then used to infect NIH3T3 target cells. EGFP expression, recloning and sequencing of integrated proviruses demonstrated the correct reassembly of the transduced ubiquitin/EGFP transcription unit in these infected cells. CONCLUSIONS: This facile and convenient system should allow production of retroviral vectors encoding potentially toxic proteins, cell cycle inhibitors or inducers of apoptosis, all of which would interfere with vector production if expressed in the retroviral packaging cell.

L2 ANSWER 3 OF 39 MEDLINE DUPLICATE 2
Full Text
AN 1999373448 MEDLINE
DN 99373448 PubMed ID: 10441560
TI Self-inactivating lentiviral vectors with **U3** and U5 modifications.
AU Iwakuma T; Cui Y; Chang L J
CS Gene Therapy Center, University of Florida, Gainesville, Florida, 32610-0266, USA.
NC HL-59412 (NHLBI)
SO VIROLOGY, (1999 Aug 15) 261 (1) 120-32.
Journal code: XEA; 0110674. ISSN: 0042-6822.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199909
ED Entered STN: 19990925
Last Updated on STN: 19990925

Entered Medline: 19990907

AB Lentiviral vectors have gained much attention in recent years mainly because they integrate into nondividing host-cell genomes. For clinical applications, a safe and efficient lentiviral vector system is required. Previously, we have established a human immunodeficiency virus type 1 (HIV-1)-derived three-plasmid lentiviral vector system for viral vector production which includes a packaging vector pHP, a transducing vector pTV, and an envelope-encoding plasmid pHEF-VSVG. Cotransfection of these three plasmids into TE671 human rhabdomyosarcoma cells routinely yields 10(5)-10(6) infectious units per milliliter in 24 h. Here we have extensively modified long terminal repeats (LTRs) of pTV to generate a safer lentiviral vector system. The 5' **U3** was replaced with a truncated cytomegalovirus (CMV) immediate early (IE) enhancer/TATA promoter and the 3' **U3** (except for the integration attachment site) was also deleted. These modifications resulted in a vector with 80% wild-type vector efficiency. Further deletion of 3' U5 impaired vector function; however, this problem was solved by replacing the 3' U5 with bovine growth hormone **polyadenylation** (bGHpA) sequence. The pTV vector containing all these modifications including the 5' promoter substitution, the 3' **U3** deletion, and the substitution of 3' U5 with bGHpA exhibited a self-inactivating (SIN) phenotype after transduction, transduced both dividing and nondividing cells at similar efficiencies, and produced vector titers twice as high as that of the wild-type construct. Thus, both safety and efficacy of the HP/TV vector have been improved by these LTR modifications. Further deletion of 5' U5 impaired vector efficiency, suggesting that the 5' U5 has critical roles in vector function.

L2 ANSWER 4 OF 39 MEDLINE DUPLICATE 3

Full Text

AN 97332385 MEDLINE

DN 97332385 PubMed ID: 9188619

TI Complete nucleotide sequence of the new simian T-lymphotropic virus, STLV-PH969 from a Hamadryas baboon, and unusual features of its long terminal repeat.

AU Van Brussel M; Goubau P; Rousseau R; Desmyter J; Vandamme A M

CS Rega Institute for Medical Research and University Hospitals, Leuven, Belgium.. vanbruss@uz.kuleuven.ac.be

SO JOURNAL OF VIROLOGY, (1997 Jul) 71 (7) 5464-72.
Journal code: KCV; 0113724. ISSN: 0022-538X.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

OS GENBANK-Y07616

EM 199707

ED Entered STN: 19970721
Last Updated on STN: 19970721
Entered Medline: 19970710

AB A third type of primate T-lymphotropic virus, PTLV-L, with STLV-PH969 as a prototype, has recently been isolated from an African baboon (Papio hamadryas). Classification of this virus has been based on partial sequence analysis of cDNA from a virus-producing cell line, PH969. We obtained the complete nucleotide sequence of this virus with a proviral genome of 8,916 bp. All major genes, homologous in all human T-cell lymphotropic virus (HTLV)-related viruses, and their corresponding mRNAs, including appropriate splicing, were identified. One additional nonhomologous open reading frame in the proximal pX region is accessible for translation through alternative splicing. Sequence comparison shows that STLV-PH969 is equidistantly related to HTLV type 1 (HTLV-1) and HTLV-2. In all coding regions, the similarity tends to be the lowest between STLV-PH969 and HTLV-1. However, in the long terminal repeat (LTR)

region, the lowest similarity was found between STLV-PH969 and HTLV-2. The U3-R and R-U5 boundaries of the STLV-PH969 LTR were experimentally determined at nucleotides 268 and 524, respectively. This 695-bp LTR is 60 and 73 bp shorter than the LTRs of HTLV-1 and HTLV-2, respectively, but its general organization is similar to the one found in the HTLV-bovine leukemia virus genus. In the long region between the **polyadenylation** signal and the poly(A) site, sequence similarity with the HTLV-1 Rex-responsive element (RexRE) core and secondary structure prediction suggest the presence of a RexRE. The presence of three 21-bp repeats is conserved within the U3 region of HTLV-1, HTLV-2, and BLV. Only two direct repeats with similarity to these Tax-responsive elements were found in the STLV-PH969 LTR, which might suggest differences in the Tax-mediated transactivation of this virus. We conclude that STLV-PH969 has all the genes and genomic regions to suggest a replication cycle comparable to that of HTLV-1 and HTLV-2.

L2 ANSWER 5 OF 39 MEDLINE DUPLICATE 4
Full Text
AN 97220003 MEDLINE
DN 97220003 PubMed ID: 9121461
TI Promoter-proximal poly(A) sites are processed efficiently, but the RNA products are unstable in the nucleus.
AU Scott J M; Imperiale M J
CS Department of Microbiology and Immunology, University of Michigan Medical School, Ann Arbor 48109-0620, USA.
NC GM34902 (NIGMS)
SO MOLECULAR AND CELLULAR BIOLOGY, (1997 Apr) 17 (4) 2127-35.
Journal code: NGY; 8109087. ISSN: 0270-7306.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199704
ED Entered STN: 19970506
Last Updated on STN: 19970506
Entered Medline: 19970424
AB The presence of two **polyadenylation** signals in the primary transcript of the human immunodeficiency virus type 1 (HIV-1) provirus leads to a requirement for regulation of 3'-end processing. To ensure that viral genome replication and gene expression occur, **polyadenylation** must occur at the poly(A) site transcribed from the 3' long terminal repeat (LTR) but not the 5' LTR. Models that have been proposed to explain this regulation include (i) inhibition of the 5' site as a result of proximity to the promoter and (ii) enhancement of the 3' site by U3 sequences that are transcribed upstream of only the 3' poly(A) site. In previous studies designed to investigate these models, a reduction in the levels of steady-state RNA was observed when the HIV-1 poly(A) site was placed within 500 nucleotides of the cap site. Although these findings were interpreted to be the result of promoter proximity effects on 3'-end processing, in vitro studies demonstrated that the HIV-1 poly(A) site was equally functional in promoter-proximal and promoter-distal positions. These results led to the hypothesis that, in vivo, the poly(A) site is fully active at this close distance but the short transcripts produced are highly unstable in the nucleus and undergo rapid degradation, precluding their appearance as abundant mRNAs in the steady-state pool. To investigate the biogenesis of these short RNAs in vivo, experiments were performed to examine directly the nuclear processing rates of the HIV-1 poly(A) site in intact cells. By using recombinant adenoviruses as expression vectors, it is now demonstrated conclusively that the HIV-1 poly(A) site is indeed processed at equivalent levels independent of its distance from the promoter. Although transcripts containing the promoter-proximal poly(A) site are processed efficiently, most undergo

degradation in the nucleus instead of nucleocytoplasmic transport.

L2 ANSWER 6 OF 39 MEDLINE DUPLICATE 5
Full Text
AN 97479470 MEDLINE
DN 97479470 PubMed ID: 9338122
TI The GT-rich sequence in the U5 region of Rous sarcoma virus long terminal repeat is required for transcription termination and 3' processing.
AU Cleavinger P J; Kandala J C; Guntaka R V
CS Department of Molecular Microbiology and Immunology, School of Medicine, University of Missouri-Columbia 65212, USA.
NC CA 54192 (NCI)
SO FOLIA BIOLOGICA, (1997) 43 (4) 153-60.
Journal code: EYH; 0234640. ISSN: 0015-5500.
CY Czech Republic
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199712
ED Entered STN: 19980109
Last Updated on STN: 19980109
Entered Medline: 19971208
AB The sequences in the LTR of Rous sarcoma virus that are required for transcription termination and **polyadenylation** have been determined. A vector containing LTR-neo-LTR has been constructed and deletions in the U5 region of the downstream LTR have been made. The DNAs from wild-type and deletion mutant recombinant plasmids were introduced into QT6 cells and G418-resistant transformants were selected. Those transformants with neo sequences in the arrangement, LTR-neo-LTR, were analyzed for transcription termination and **polyadenylation** by Northern blot analysis and by S1 protection experiments. The results indicate that the **polyadenylation** signal, AATAAA, located in the **U3** region alone, is not sufficient for 3' end processing and that the sequence between +20 and +44 in the U5 region is absolutely required for transcription termination or endonucleolytic cleavage and **polyadenylation**.

L2 ANSWER 7 OF 39 MEDLINE DUPLICATE 6
Full Text
AN 96190559 MEDLINE
DN 96190559 PubMed ID: 8627681
TI A common mechanism for the enhancement of mRNA 3' processing by **U3** sequences in two distantly related lentiviruses.
AU Graveley B R; Gilmartin G M
CS Department of Microbiology and Molecular Genetics, University of Vermont, Burlington 05405, USA.
NC GM46624 (NIGMS)
SO JOURNAL OF VIROLOGY, (1996 Mar) 70 (3) 1612-7.
Journal code: KCV; 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199606
ED Entered STN: 19960708
Last Updated on STN: 19970203
Entered Medline: 19960627
AB The protein coding regions of all retroviral pre-mRNAs are flanked by a direct repeat of R-U5 sequences. In many retroviruses, the R-U5 repeat contains a complete core poly(A) site-composed of a highly conserved AAUAAA hexamer and a GU-rich downstream element. A mechanism that allows for the bypass of the 5' core poly(A) site and the exclusive use of the 3' core poly(A) site must therefore exist. In human immunodeficiency virus

type 1 (HIV-1), sequences within the U3 region appear to play a key role in poly(A) site selection. U3 sequences are required for efficient 3' processing at the HIV-1 poly(A) site both in vivo and in vitro. These sequences serve to promote the interaction of cleavage and **polyadenylation** specificity factor (CPSF) with the core poly(A) site. We have now demonstrated the presence of a functionally analogous 3' processing enhancer within the U3 region of a distantly related lentivirus, equine infectious anemia virus (EIAV). U3 sequences enhanced the processing of the EIAV core poly(A) site sevenfold in vitro. The U3 sequences also enhanced the stability of CPSF binding at the core poly(A) site. Optimal processing required the TAR RNA secondary structure that resides within the R region 28 nucleotides upstream of the AAUAAA hexamer. Disruption of TAR reduced processing, while compensatory changes that restored the RNA structure also restored processing to the wild-type level, suggesting a position dependence of the U3-encoded enhancer sequences. Finally, the reciprocal exchange of the EIAV and HIV U3 regions demonstrated the ability of each of these sequences to enhance both 3' processing and the binding of CPSF in the context of the heterologous core poly(A) site. The impact of U3 sequences upon the interaction of CPSF at the core poly(A) site may therefore represent a common strategy for retroviral poly(A) site selection.

L2 ANSWER 8 OF 39 MEDLINE DUPLICATE 7
Full Text
AN 94309138 MEDLINE
DN 94309138 PubMed ID: 8035477
TI Functional and biological properties of an avian variant long terminal repeat containing multiple A to G conversions in the U3 sequence.
AU Felder M P; Laugier D; Yatsula B; Dezelee P; Calothy G; Marx M
CS Unite de Recherche Associee 1443 du Centre National de la Recherche Scientifique, Institut Curie, Centre Universitaire, Orsay, France.
SO JOURNAL OF VIROLOGY, (1994 Aug) 68 (8) 4759-67.
Journal code: KCV; 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-X77628; GENBANK-X77629; GENBANK-X77630
EM 199408
ED Entered STN: 19940825
Last Updated on STN: 19980206
Entered Medline: 19940815
AB We previously reported that infection of chicken embryonic neuroretina cells with Rous-associated virus type 1 leads to the frequent occurrence of spliced readthrough transcripts containing viral and cellular sequences. Generation of such chimeric transcripts constitutes a very early step in oncogene transduction. We report, here, the isolation of a c-mil transducing retrovirus, designated IC4, which contains a highly mutated U3 sequence in which 48% of A is converted to G. Functional analysis of this variant U3 indicated that these mutations do not impair viral transcription and replication; however, they abolish functioning of its **polyadenylation** signal, thus allowing readthrough transcription of downstream cellular sequences. On the basis of these results, we designed a nonreplicative retroviral vector, pIC4Neo, expressing the neomycin resistance (Neo(r)) gene under the control of the IC4 long terminal repeat. Infection of nondividing neuroretina cells with virus produced by a packaging cell line transfected with pIC4Neo occasionally resulted in sustained cell proliferation. Two independent G418-resistant proliferating cultures were found to express hybrid RNAs containing viral and cellular sequences. These sequences were characterized by reverse transcription-PCR and were identified in both cultures, suggesting that proliferation was correlated with a common integration locus. These results indicate that

IC4Neo virus functions as a useful insertional mutagen and may allow identification of genes potentially involved in regulation of cell division.

L2 ANSWER 9 OF 39 MEDLINE DUPLICATE 8
Full Text
AN 94173734 MEDLINE
DN 94173734 PubMed ID: 8127714
TI Binding of nuclear factors to a satellite DNA of retroviral origin with marked differences in copy number among species of the rodent Ctenomys.
AU Pesce C G; Rossi M S; Muro A F; Reig O A; Zorzopulos J; Kornblihtt A R
CS Instituto de Investigaciones en Ingenieria Genetica y Biologia Molecular (INGEBI-CONICET), Buenos Aires, Argentina.
SO NUCLEIC ACIDS RESEARCH, (1994 Feb 25) 22 (4) 656-61.
Journal code: O8L; 0411011. ISSN: 0305-1048.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199404
ED Entered STN: 19940420
Last Updated on STN: 19940420
Entered Medline: 19940411
AB The major satellite DNA of the subterranean rodent Ctenomys, named RPCS, contains several consensus sequences characteristic of the U3 region of retroviral long terminal repeats (LTRs), such as a polypurine tract, CCAAT boxes, binding sites for the CCAAT/enhancer-binding protein (C/EBP), a TATA box and putative **polyadenylation** signals. RPCS presents an enormous variation in abundance between species of the same genus: while C. australis or C. talarum have approximately 3 x 10(6) copies per genome, C. opimus has none. A sequence (RPCS-I) with identity to the SV40-enhancer core element, present in all the repeating units of the satellite is specifically protected in DNase I footprintings. Competitions of band-shift assays with different transcription factor binding sites indicate that binding to RPCS-I is specific and involves CCAAT proteins related to NF-1, but not to C/EBP. By the use of quantitative protein/DNA binding assays we determined that, despite of their conspicuous difference in RPCS copy number, C. talarum and C. opimus have equivalent amounts and identical quality of RPCS-binding proteins. These results are consistent with the observation, by in situ hybridization, that RPCS is clustered in heterochromatic regions, where it might have restricted accessibility to transcription factors in vivo. This is the first report of the binding of transcription factors to a satellite DNA of retroviral origin.

L2 ANSWER 10 OF 39 MEDLINE DUPLICATE 9
Full Text
AN 94147991 MEDLINE
DN 94147991 PubMed ID: 8313890
TI Poly(A) site selection in the yeast Ty retroelement requires an upstream region and sequence-specific titratable factor(s) in vitro.
AU Hou W; Russnak R; Platt T
CS Department of Biochemistry, University of Rochester Medical Center, NY 14642.
NC 2-R01-GM35658 (NIGMS)
SO EMBO JOURNAL, (1994 Jan 15) 13 (2) 446-52.
Journal code: EMB; 8208664. ISSN: 0261-4189.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199403
ED Entered STN: 19940330

Last Updated on STN: 19990129
Entered Medline: 19940321

AB In the Ty retrotransposon of Saccharomyces cerevisiae, as in most retroelements, the **polyadenylation** site of the 5' long terminal repeat (LTR) is ignored and the one in the 3' LTR is efficiently used. We examine here the contribution to this poly(A) site selection of the region termed '**U3**', corresponding to the upstream non-transcribed portion of the 5' LTR. Using an established assay in vitro, we find that 3' processing is accurate and efficient with an RNA substrate corresponding to most of the LTR, whereas none is detectable with a shorter transcript lacking the **U3** region, thus explaining why the 5' poly(A) site is ignored in genomic Ty mRNA. When HIS4 coding RNA, representing 'non-specific' sequence, replaces the **U3** region, the Ty **polyadenylation** site is activated to 50% of the wild-type level. Within one specific region (TS1) in **U3**, 90-95 nt upstream of the poly(A) site, the change of UAGUAU to UCGCAU reduces processing efficiency by half, to the non-specific level provided by other sequences or by a deletion of the TS1 region. Another region (TS2) near the poly(A) site appears to be independently responsible for the remaining half of the processing activity. Alteration of both TS1 and TS2 eliminates processing entirely. In competition assays, excess unlabeled **U3**, but not its mutated counterparts, reduces the processing of radiolabeled Ty mRNA, suggesting the involvement of some sequence-specific titratable factor(s) in the whole cell extract for **U3**-specific activation.(ABSTRACT TRUNCATED AT 250 WORDS)

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L2 ANSWER 11 OF 39 MEDLINE
Full Text
AN 94066912 MEDLINE
DN 94066912 PubMed ID: 7902524
TI Transcription termination and **polyadenylation** in retroviruses.
AU Guntaka R V
CS Department of Molecular Microbiology and Immunology, School of Medicine, University of Missouri-Columbia 65212.
SO MICROBIOLOGICAL REVIEWS, (1993 Sep) 57 (3) 511-21. Ref: 108
Journal code: M9M; 7806086. ISSN: 0146-0749.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals
EM 199401
ED Entered STN: 19940201
Last Updated on STN: 19950206
Entered Medline: 19940103

AB The provirus structure of retroviruses is bracketed by long terminal repeats (LTRs). The two LTRs (5' and 3') are identical in nucleotide sequence and organization. They contain signals for transcription initiation as well as termination and cleavage **polyadenylation**. As in eukaryotic pre-mRNAs, the two common signals, the **polyadenylation** signal, AAUAAA, or a variant AGUAAA, and the G+U-rich sequence are present in all retroviruses. However, the AAUAAA sequence is present in the **U3** region in some retroviruses and in the R region in other retroviruses. As

in animal cell RNAs, both AAUAAA and G+U-rich sequences apparently contribute to the 3'-end processing of retroviral RNAs. In addition, at least in a few cases examined, the sequences in the U3 region determine the efficiency of 3'-end processing. In retroviruses in which the AAUAAA is localized in the R region, the poly(A) signal in the 3' LTR but not the 5' LTR must be selectively used for the production of genomic RNA. It appears that the short distance between the 5' cap site and **polyadenylation** signal in the 5' LTR precludes premature termination and **polyadenylation**. Since 5' and 3' LTRs are identical in sequence and structural organization yet function differently, it is speculated that flanking cellular DNA sequences, chromatin structure, and binding of transcription factors may be involved in the functional divergence of 5' and 3' LTRs of retroviruses.

L2 ANSWER 12 OF 39 MEDLINE
Full Text
AN 94033623 MEDLINE
DN 94033623 PubMed ID: 8219281
TI Retroviral-like features in the monomer of the major satellite DNA from the South American rodents of the genus Ctenomys.
AU Rossi M S; Pesce C G; Reig O A; Kornblihtt A R; Zorzopulos J
CS GIBE, Department of Biology, FCEyN, University of Buenos Aires, Argentina.
SO DNA SEQUENCE, (1993) 3 (6) 379-81.
Journal code: A9H; 9107800. ISSN: 1042-5179.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-X69517
EM 199312
ED Entered STN: 19940117
Last Updated on STN: 19940117
Entered Medline: 19931222
AB It is well known that uninfected mammalian cells contain DNA sequences which are closely related to retroviral genomic segments. However, these sequences seldom (if ever) have been found associated to highly repetitive (satellite) DNA. RPCS is a 348 bp monomer of a major satellite DNA from the South American rodents of the genus Ctenomys. It was found that RPCS contains several elements which are typical of the U3 region of retroviral LTRs. These elements are: a) a polypurine tract; b) two enhancer core sequences; c) two NF1 binding sites; d) two C/EBP binding sites; e) two CCAAT-motifs; f) a TATA box, and g) two putative **polyadenylation** motifs. Furthermore, the relative positions of these elements are as in the U3 retroviral regions.

L2 ANSWER 13 OF 39 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
Full Text
AN 1993:81233 BIOSIS
DN PREV199395045733
TI Relative roles of signals upstream of AAUAAA and promoter proximity in regulation of human immunodeficiency virus type 1 mRNA 3' end formation.
AU Dezazzo, James D.; Scott, Jeannine M.; Imperiale, Michael J. (1)
CS (1) Dep. Microbiol. Immunology, University Michigan Med. Sch., Ann Arbor, Michigan 48109-0620
SO Molecular and Cellular Biology, (1992) Vol. 12, No. 12, pp. 5555-5562.
ISSN: 0270-7306.
DT Article
LA English
AB At least two mechanisms have been implicated in regulating poly(A) site use in human immunodeficiency virus type 1 (HIV-1): inhibition of basal signals within 500 nucleotides (nt) of the cap site, leading to specific suppression of the 5' poly(A) site, and stimulation of basal signals by

long terminal repeat U3 sequences, leading to specific activation of the 3' poly(A) site. We determined the relative contributions of these mechanisms in a HeLa cell transcription/processing reaction and by transient transfection analysis. In vitro, the efficiency of basal signals is equivalent close to (270 nt) and far from (1,080 nt) the promoter and is stimulated at least 30-fold in both positions by upstream U3 sequences. In vivo, U3 sequences also enhance processing at both positions. There are two additional effects when the poly(A) site is close to the cap site: at least a 15-fold reduction in total RNA levels and a 5-fold decrease in relative levels of RNA processed at the HIV-1 site in constructs containing U3. Both effects are overcome by insertion of upstream splicing signals in an orientation-dependent manner. Splicing appears to influence poly(A)+ RNA levels by two distinct mechanisms: stabilizing nuclear transcripts and directly stimulating 3' end formation. It is proposed that upstream elements play major roles in regulating poly(A) site choice and in controlling the subsequent fate of polyadenylated RNA. The impact of these findings on mechanisms of mRNA biogenesis in the HIV-1 provirus is discussed.

L2 ANSWER 14 OF 39 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
Full Text
AN 1993:73752 BIOSIS
DN PREV199395038252
TI Activation of HIV-1 pre-mRNA 3' processing in vitro requires both an upstream element and TAR.
AU Gilmartin, Gregory M.; Fleming, Elizabeth S.; Oetjen, Joyce
CS Dep. Microbiol. Molecular Genetics, Univ. Vermont, Given Building, Burlington, VT 05405 USA
SO EMBO (European Molecular Biology Organization) Journal, (1992) Vol. 11, No. 12, pp. 4419-4428.
ISSN: 0261-4189.
DT Article
LA English
AB The architecture of the human immunodeficiency virus type 1 (HIV-1) genome presents an intriguing dilemma for the 3' processing of viral transcripts sbd to disregard a canonical 'core' poly(A) site processing signal present at the 5' end of the transcript and yet to utilize efficiently an identical signal that residues at the 3' end of the message. The choice of processing sites of HIV-1 appears to be influenced by two factors: (i) proximity to the cap site, and (ii) sequences upstream of the core poly(A) site. We now demonstrate that an in vivo-defined upstream element that resides within the U3 region, 76 nucleotides upstream of the AAUAAA hexamer, acts specifically to enhance 3' processing at the HIV-1 core poly(A) site in vitro. We furthermore show that efficient in vitro 3' processing requires the RNA stem-loop structure of TAR, which serves to juxtapose spatially the upstream element and the core poly(A) site. An analysis of the stability of 3' processing complexes formed at the HIV-1 poly(A) site in vitro suggests that the upstream element may function by increasing processing complex stability at the core poly(A) site.

L2 ANSWER 15 OF 39 MEDLINE DUPLICATE 10
Full Text
AN 92375036 MEDLINE
DN 92375036 PubMed ID: 1508176
TI Elements upstream of the AAUAAA within the human immunodeficiency virus **polyadenylation** signal are required for efficient **polyadenylation** in vitro.
AU Valsamakis A; Schek N; Alwine J C
CS Department of Microbiology, School of Medicine, University of Pennsylvania, Philadelphia 19104-6142.
NC GM45773 (NIGMS)
SO MOLECULAR AND CELLULAR BIOLOGY, (1992 Sep) 12 (9) 3699-705.

Journal code: NGY; 8109087. ISSN: 0270-7306.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199209
ED Entered STN: 19921009
Last Updated on STN: 19970203
Entered Medline: 19920923
AB Recent in vivo studies have identified specific sequences between 56 and 93 nucleotides upstream of a **polyadenylation** [poly(A)] consensus sequence, AAUAAA, in human immunodeficiency virus type 1 (HIV-1) that affect the efficiency of 3'-end processing at this site (A. Valsamakis, S. Zeichner, S. Carswell, and J. C. Alwine, Proc. Natl. Acad. Sci. USA 88:2108-2112, 1991). We have used HeLa cell nuclear extracts and precursor RNAs bearing the HIV-1 poly(A) signal to study the role of upstream sequences in vitro. Precursor RNAs containing the HIV-1 AAUAAA and necessary upstream (**U3** region) and downstream (U5 region) sequences directed accurate cleavage and **polyadenylation** in vitro. The in vitro requirement for upstream sequences was demonstrated by using deletion and linker substitution mutations. The data showed that sequences between 56 and 93 nucleotides upstream of AAUAAA, which were required for efficient **polyadenylation** in vivo, were also required for efficient cleavage and **polyadenylation** in vitro. This is the first demonstration of the function of upstream sequences in vitro. Previous in vivo studies suggested that efficient **polyadenylation** at the HIV-1 poly(A) signal requires a spacing of at least 250 nucleotides between the 5' cap site and the AAUAAA. Our in vitro analyses indicated that a precursor containing the defined upstream and downstream sequences was efficiently cleaved at the **polyadenylation** site when the distance between the 5' cap and the AAUAAA was reduced to at least 140 nucleotides, which is less than the distance predicted from in vivo studies. This cleavage was dependent on the presence of the upstream element.

L2 ANSWER 16 OF 39 MEDLINE DUPLICATE 11
Full Text
AN 92148961 MEDLINE
DN 92148961 PubMed ID: 1310773
TI ART-CH, a new chicken retroviruslike element.
AU Gudkov A V; Komarova E A; Nikiforov M A; Zaitsevskaya T E
CS Laboratory of Molecular Genetics, Cancer Research Center, Moscow, Russia.
SO JOURNAL OF VIROLOGY, (1992 Mar) 66 (3) 1726-36.
Journal code: KCV; 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-M85057; GENBANK-M85058
EM 199203
ED Entered STN: 19920405
Last Updated on STN: 19990129
Entered Medline: 19920316
AB A 3' region of a previously unknown retroviruslike element named ART-CH (avian retrotransposon from chicken genome) was obtained in the course of polymerase chain reaction-mediated cloning of avian leukosis virus long terminal repeats (LTRs) from DNAs of infected chicken cells. About 50 copies of ART-CH are present in the genome of chickens of different breeds. ART-CH is not found in DNA of quails, ducks, turkeys, or several other birds tested. The ART-CH element is about 3 kb in size, including 388 bp LTRs. The major class of ART-CH-specific RNA, also 3 kb in size, is detected in various organs of chickens. An ART-CH polypurine tract, a tRNA(Trp)-binding site, regions around the TATA box and **polyadenylation**

signal, and the beginning of the putative gag gene strongly resemble the corresponding regions of avian leukosis viruses and EAV, the two described classes of chicken retroviruses. An open reading frame capable of encoding a polypeptide with a putative transmembrane domain is located upstream of the right ART-CH LTR. This sequence, as well as the **U3** and U5 regions of the ART-CH LTR, has no obvious similarities with the corresponding parts of other known vertebrate retroviruses and retrotransposons. A short sequence upstream of the right LTR of ART-CH is very similar to sequences which flank the 3' ends of the oncogenes v-src, v-myc, v-fps, and v-crk in four different recombinant avian retroviruses and which are absent from the genomes of other studied avian retroviruses. Thus, ART-CH is a new endogenous chicken provirus that may participate in the formation of recombinant oncogenic retroviruses.

L2 ANSWER 17 OF 39 MEDLINE DUPLICATE 12
Full Text
AN 92224890 MEDLINE
DN 92224890 PubMed ID: 1373376
TI Regulation of **polyadenylation** in human immunodeficiency virus (HIV): contributions of promoter proximity and upstream sequences.
AU Cherrington J; Ganem D
CS Howard Hughes Medical Institute, Department of Microbiology, University of California Medical Center, San Francisco 94143-0502.
SO EMBO JOURNAL, (1992 Apr) 11 (4) 1513-24.
Journal code: EMB; 8208664. ISSN: 0261-4189.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199205
ED Entered STN: 19920607
Last Updated on STN: 19970203
Entered Medline: 19920515
AB Retroviruses synthesize a terminally redundant genomic RNA that contains canonical **polyadenylation** signals at both ends. Production of this RNA requires that the 5' copy of these signals be ignored, while the 3' copy must be utilized. Two models have been presented for how this occurs in the human immunodeficiency virus, HIV: (i) the core HIV poly(A) signals (AAUAAA and a downstream GU-rich element) might be inefficient and require supplementation by activating sequences found only at the 3' end of the RNA; or (ii) cap site proximity might actively suppress **polyadenylation** at the 5' site. We have examined both possibilities in HIV-infected cells and in cells transfected with a variety of model constructs. We find that infected cells harbor few or no detectable products of 5' **polyadenylation**; however, the core HIV processing signals can mediate processing fairly efficiently (65%) when positioned at the 3' end of heterologous transcripts. While this efficiency can be further increased to greater than 95% by inclusion of upstream sequences from the viral **U3** region, the absence of these **U3** signals is insufficient by itself to account for 5' signal bypass. By contrast, the efficiency of these core elements is greatly suppressed when they are positioned within approximately 450 nucleotides of the cap site. This distance-related suppression can be modestly diminished by insertion of **U3** sequences between the cap site and HIV poly(A) signal. We suggest that the primary determinant of 5' poly(A) site bypass is cap site proximity; the absence of **U3** sequences at this position contributes secondarily to the bypass by enhancing the sensitivity of the pA signal to the suppressive effects of promoter proximity.

L2 ANSWER 18 OF 39 MEDLINE DUPLICATE 13
Full Text
AN 93033143 MEDLINE

DN 93033143 PubMed ID: 1413518
TI Structural and functional organization of the human endogenous retroviral ERV9 sequences.
AU Lania L; Di Cristofano A; Strazzullo M; Pengue G; Majello B; La Mantia G
CS Dipartimento di Genetica, Biologia Generale e Molecolare University of Naples, Italy.
SO VIROLOGY, (1992 Nov) 191 (1) 464-8.
Journal code: XEA; 0110674. ISSN: 0042-6822.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-M92647; GENBANK-M92648
EM 199211
ED Entered STN: 19930122
Last Updated on STN: 19970203
Entered Medline: 19921116
AB The human genome contains a variety of genetic elements similar in structure to retroviruses and retrotransposons. We report here the structural and functional organization of a novel human endogenous retroviral family (ERV9). Three polyadenylated RNAs, 8, 2, and 1.5 kb long, are detected by Northern blot in undifferentiated embryonal carcinoma NT2/D1 cells. Upon genomic cloning of an expressed ERV9 locus, we demonstrated that the three polyadenylated RNAs are originated by a single ERV9 locus by alternative usage of splicing and **polyadenylation** signals. DNA sequence analysis of different ERV9 LTRs have revealed that they are heterogeneous in length and that the length variability is due to the number of tandemly repeated subelements present in both U3 and U5 regions; moreover, the ERV9 LTRs are capable to drive expression of a reporter gene in transient expression assays. Finally, analysis of the ERV9 5' transcription start site has allowed us to define the U3-R-U5 organization of the ERV9 LTR.

L2 ANSWER 19 OF 39 MEDLINE DUPLICATE 14
Full Text
AN 92223097 MEDLINE
DN 92223097 PubMed ID: 1562598
TI Sequence variations and promoter activities of long terminal repeats from rat intracisternal A-particles.
AU Furter C S; Rentsch J M; Bertchtold M W
CS Institute of Pharmacology and Biochemistry, University of Zurich, Switzerland.
SO BIOCHIMICA ET BIOPHYSICA ACTA, (1992 Mar 24) 1130 (2) 213-7.
Journal code: A0W; 0217513. ISSN: 0006-3002.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-M55184; GENBANK-M55185; GENBANK-M55186; GENBANK-M55187; GENBANK-M55188; GENBANK-M91426; GENBANK-M91427; GENBANK-M91428; GENBANK-M91429; GENBANK-M91430
EM 199205
ED Entered STN: 19920607
Last Updated on STN: 19970203
Entered Medline: 19920521
AB Nucleotide sequences of three novel rat long terminal repeats (LTR) of intracisternal A-particles (IAP) were determined and compared with two previously published solitary rat IAP LTRs from the genomic clone H12 (Furter et al. (1989) J. Biol. Chem. 264, 18276-18279) and from the upstream region of the oncomodulin (OM) gene (Banville and Boie (1989) J. Mol. Biol. 207, 481-490). These five LTRs have a length of 286 to 370 bp and show the major variability within the U3 region. The CCAAT and the

TATA boxes, the AATAAA **polyadenylation** signals and the CA **polyadenylation** sites are well conserved in sequence and position in all five LTRs, whereas several putative transcriptional factor binding sites in the **U3** domain show considerable heterogeneity. The transcriptional activities of three LTRs were tested in transient gene expression assays using the human growth hormone (hGH) reporter gene in chemically transformed T14c cells which produce considerable amounts of oncomodulin. Promoter strengths of the three investigated LTRs varied considerably.

L2 ANSWER 20 OF 39 MEDLINE DUPLICATE 15
Full Text
AN 92093601 MEDLINE
DN 92093601 PubMed ID: 1754382
TI Regulation of **polyadenylation** in hepatitis B viruses: stimulation by the upstream activating signal PS1 is orientation-dependent, distance-independent, and additive.
AU Russnak R H
CS Department of Biochemistry, University of Rochester Medical Center, NY 14642.
SO NUCLEIC ACIDS RESEARCH, (1991 Dec 11) 19 (23) 6449-56.
Journal code: O8L; 0411011. ISSN: 0305-1048.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199201
ED Entered STN: 19920216
Last Updated on STN: 19920216
Entered Medline: 19920129
AB Hepatitis B viruses replicate by reverse transcription of a genomic RNA which harbors terminal redundancies. The synthesis of this RNA requires that transcription proceed twice through the **polyadenylation** (pA) site which, in mammalian strains, is flanked by the variant hexanucleotide UAUAAA and a T-rich downstream domain. These core elements are by themselves virtually defective in 3' end processing and require multiple upstream accessory elements which regulate pA site use. In ground squirrel hepatitis B virus (GSHV), one of these signals (PS1; -215 to -107 relative to UAUAAA) is transcribed only at the 3' end of genomic RNA and as such is analogous to retroviral **U3** sequences. PS1 cooperates with other signals to enhance pA site use to very high levels and can be further sub-divided into two regions (A and B) which contribute equally to 3' end processing. Critical residues within PS1B have been localized to a 15 bp A/T-rich stretch which displays homology to other known upstream activating signals. A 15 bp segment within PS1A which has the identical A/T content but a divergent primary sequence plays a diminished role in processing. Furthermore, PS1 can activate GSHV core element usage autonomously. This stimulation has been shown to be additive since multiple copies of PS1 progressively increase **polyadenylation**, a phenomenon which also demands that PS1 exert its influence from a variety of distances from the hexanucleotide signal.

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L3            8 PROMOTER CONVERSION

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DUPLICATE PREFERENCE IS 'MEDLINE, BIOSIS'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L3
L4            4 DUPLICATE REMOVE L3 (4 DUPLICATES REMOVED)

=> d 1-4 bib ab
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L4 ANSWER 1 OF 4 MEDLINE DUPLICATE 1
Full Text
AN 1998292750 MEDLINE
DN 98292750 PubMed ID: 9620967
TI OpaR, a homolog of Vibrio harveyi LuxR, controls opacity of Vibrio parahaemolyticus.
AU McCarter L L
CS Department of Microbiology, University of Iowa, Iowa City 52242, USA.. linda-mccarter@uiowa.edu
NC GM43196 (NIGMS)
SO JOURNAL OF BACTERIOLOGY, (1998 Jun) 180 (12) 3166-73.
Journal code: HH3; 2985120R. ISSN: 0021-9193.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AF035967
EM 199807
ED Entered STN: 19980716
Last Updated on STN: 20000303
Entered Medline: 19980706
AB Vibrio parahaemolyticus is an organism well adapted to communal life on surfaces. When grown on a surface or in a viscous layer, the bacterium induces a large gene system and differentiates to swarmer cells capable of movement over and colonization of surfaces. V. parahaemolyticus displays additional phenotypic versatility manifested as variable colony morphology, switching between translucent and opaque colony types. Although not itself luminescent, V. parahaemolyticus produces autoinducer molecules capable of inducing luminescence in Vibrio harveyi. To examine the role of quorum signaling in the lifestyles of V. parahaemolyticus, the functional homolog of the gene encoding the V. harveyi autoinducer-controlled transcriptional regulatory protein LuxR was cloned. Sequence analysis of the clone predicted an open reading frame with a deduced product 96% identical to LuxR. Introduction of the clone carrying the luxR-like locus into V. parahaemolyticus dramatically affected colony morphology, converting a translucent strain to an opaque one. When the coding sequence for the luxR homolog was placed under the control of the Ptac **promoter**, **conversion** to the opaque phenotype became inducible by isopropyl-beta-D-thiogalactopyranoside. Allelic disruption of the luxR-like gene on the chromosome of an opaque strain produced a translucent strain proficient in swarming ability. Primer extension mapping demonstrated opaR transcription in opaque but not translucent cell types. It is postulated that this gene, which has been named opaR, encodes a transcription factor controlling cell type. The underlying genetic basis for opaque-translucent variation may be the consequence of a genomic alteration detected in the opaR locus of opaque and translucent strains.

L4 ANSWER 2 OF 4 MEDLINE DUPLICATE 2
Full Text
AN 1998088888 MEDLINE
DN 98088888 PubMed ID: 9428612
TI Inducible expression of p21WAF-1/CIP-1/SDI-1 from a **promoter conversion** retroviral vector.
AU Mrochen S; Klein D; Nikol S; Smith J R; Salmons B; Gunzburg W H
CS Institute of Molecular Virology, GSF-National Research Center for Environment and Health, Universitat Munchen, Germany.
SO JOURNAL OF MOLECULAR MEDICINE, (1997 Nov-Dec) 75 (11-12) 820-8.
Journal code: B8C; 9504370. ISSN: 0946-2716.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English

FS Priority Journals
EM 199802
ED Entered STN: 19980224
Last Updated on STN: 19980224
Entered Medline: 19980209
AB Constitutive, high-level expression of the potentially therapeutic WAF-1/CIP-1/SDI-1 gene is incompatible with cell growth. A **promoter conversion** retroviral vector carrying the WAF-1/CIP-1/SDI-1 gene under the transcriptional control of the glucocorticoid inducible promoter of mouse mammary tumor virus was used to infect human bladder carcinoma or feline kidney cells. Reduced cell growth due to a greater proportion of cells being in the G0/G1 phase of the cell cycle was observed when WAF-1/CIP-1/SDI-1 expression was activated by addition of glucocorticoid hormone. This system demonstrates the potential long-term therapeutic use of WAF-1/CIP-1/SDI-1 delivered by retroviral vectors for inhibiting the growth of rapidly proliferating cells. Moreover, the conditional expression of genes such as WAF-1/CIP-1/SDI-1 from such retroviral vectors may facilitate analysis of their function.

L4 ANSWER 3 OF 4 MEDLINE DUPLICATE 3
Full Text
AN 96228295 MEDLINE
DN 96228295 PubMed ID: 8642278
TI Importance of low affinity Elf-1 sites in the regulation of lymphoid-specific inducible gene expression.
AU John S; Marais R; Child R; Light Y; Leonard W J
CS Laboratory of Molecular Immunology, National Heart, Lung, and Blood Institute, National Institutes of Health, Bethesda, Maryland 20892, USA.
SO JOURNAL OF EXPERIMENTAL MEDICINE, (1996 Mar 1) 183 (3) 743-50.
Journal code: I2V; 2985109R. ISSN: 0022-1007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199607
ED Entered STN: 19960726
Last Updated on STN: 19970203
Entered Medline: 19960716
AB Elf-1 is an Ets family transcription factor that regulates a number of inducible lymphoid-specific genes, including those encoding interleukin 3 (IL-3), granulocyte/macrophage colony-stimulating factor (GM-CSF), and the IL-2 receptor (IL-2R) alpha chain. A minimal oligonucleotide spanning the IL-2R alpha Elf-1 site (-97/-84) bound Elf-1 poorly, but binding activity markedly increased when this oligonucleotide was multimerized or flanking sequences were added. This result is consistent with the requirement of accessory proteins for efficient Elf-1 binding, as has been demonstrated for the GM-CSF and IL-3 promoters. A binding site selection analysis revealed the optimal Elf-1 consensus motif to be A(A/t)(C/a)CCGGAAGT(A/S), which is similar to the consensus motif for the related Drosophila E74 protein. This minimal high affinity site could bind Elf-1 and functioned as a stronger transcription element than the -97/-84 IL-2R alpha oligonucleotide when cloned upstream of a heterologous promoter. In contrast, in the context of the IL-2R alpha **promoter, conversion** of the naturally occurring low affinity Elf-1 site to an optimal site decreased inducible activation of a reporter construct in Jurkat cells. This finding may be explained by the observation that another Ets family protein, ER GB/Fli-1, can efficiently bind only to the optimal site, and in this context, interferes with Elf-1 binding. Therefore, high affinity Elf-1 sites may lack sufficient binding specificity, whereas naturally occurring low affinity sites presumably favor the association of Elf-1 in the context of accessory proteins. These findings offer an explanation for the lack of optimal sites in any of the known Elf-1-regulated genes.

L4 ANSWER 4 OF 4 MEDLINE DUPLICATE 4
Full Text
AN 93113085 MEDLINE
DN 93113085 PubMed ID: 1369072
TI Conversion of dethiobiotin to biotin in cell-free extracts of Escherichia coli.
AU Ifuku O; Kishimoto J; Haze S; Yanagi M; Fukushima S
CS Shiseido Research Center, Yokohama, Japan.
SO BIOSCIENCE, BIOTECHNOLOGY, AND BIOCHEMISTRY, (1992 Nov) 56 (11) 1780-5.
Journal code: BDP; 9205717. ISSN: 0916-8451.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS B
EM 199302
ED Entered STN: 19950809
Last Updated on STN: 19950809
Entered Medline: 19930203
AB We constructed the plasmid pTTB151 in which the E. coli bioB gene was expressed under the control of the tac **promoter**. **Conversion** of dethiobiotin to biotin was demonstrated in cell-free extracts of E. coli carrying this plasmid. The requirements for this biotin-forming reaction included fructose-1,6-bisphosphate, Fe2+, S-adenosyl-L-methionine, NADPH, and KCl, as well as dethiobiotin as the substrate. The enzymes were partially purified from cell-free extracts by a procedure involving ammonium sulfate fractionation. Our results suggest that an unidentified enzyme(s) besides the bioB gene product is obligatory for the conversion of dethiobiotin to biotin.

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	21.57	21.78

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